tion carcinoma) 1 each from pulmonary metastasis and pleural fluid. All showed several chromosomal abnormalities. With the modal number varying between 56 - 87 chromosomes. The consistent finding in 9/10 cases was a deletion of 3p while 1 had a translocation involving chromosome 3.

In this study tumors irrespective of being a NSCLC or SCLC showed that chromosome 3 was consistently involve hinting that this cytogenetic profile could evolve as a new diagnostic tool in difficult clinical situations.

P3-005 **BSTB: Molecular Pathology Posters, Wed, Sept 5 – Thur, Sept 6**

The tissue array analysis of the aberrant expression of HLA class I molecules in human non-small cell lung cancer

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Background: Down-regulation or loss of human leukocyte antigen (HLA) class I has been reported in various malignancies in association with poor patient survival. These findings may reflect an escape of tumor cells losing intact HLA class I molecules from HLA class I-restricted, tumor-associated antigen-specific cytotoxic T lymphocytes in recognition as well as in destruction. However, clinical impact of the down-regulation or loss of HLA class I in patients with non-small cell lung cancer (NSCLC) is still unknown. In the present study, we investigated clinical significance of alteration in expression of HLA class I molecules in surgically resected NSCLC using tissue array analysis.

Method: Formalin-fixed paraffin-embedded sections were obtained from 105 NSCLC (60 adenocarcinoma, 37 squamous cell carcinoma, and 6 large cell carcinoma). HLA class I and β2- microglobulin expression were analyzed using tissue array analysis. Imuunohistochemical study was performed using anti-HLA class I mAb EMR8-5 and anti-β2- microglobulin mAb BBM1/sc-13565. Correlation between HLA class I or β2- microglobulin expression and clinicopathological parameter was analyzed statistically. Additionally, survival analysis was also performed using Kaplan-Meier's method.

Result: There was no significant correlation between HLA class I and clinicopathological parameters. On the other hand, significant correlations were observed between β2- microglobulin expression and tumor size(p=0.0003), histological subtype(p=0.0285), nodal status(p=0.0335),and pathological stage(p=0.0335). Loss of β2- microglobulin expression was correlated with poor patient survival (p=0.0121), although HLA class I was not(p=0.8243).

Conclusion: Although clinical impact of HLA alteration was still unclear, β2- microglobulin expression might be involved in cancer progression in human NSCLC possibly through aberration of several molecules which contains β2- microglobulin.

P3-006 **BSTB: Molecular Pathology Posters, Wed, Sept 5 – Thur, Sept 6**

Rtk signal pathways differentially activated in lung adenocarcinoma

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Purpose: To assess the activation patterns of RTK pathways, i.e. protein kinase B (PKB/Akt) and mitogen activated protein kinase (MAPK) pathways, in lung adenocarcinoma as compared with clinico-pathological factors and status of EGFR and KRAS mutations.

Experimental Design: 99 evaluable cases with primary lung adenocarcinomas surgically resected between 1998 and 1999 were selected for this study. Akt and MAPK phosphorylation (pAkt and pMAPK) was examined by means of immunohistochemistry. We defined that cases with pAkt+/pMAPK- immunoreactivity to be the "Akt pathway type" and the pAkt-/pMAPK+ pattern to be the "MAPK pathway type." Both positive and negative controls were prepared using mouse xenografts of PC-3 (pAkt+/MAPK-) and HTB26 (pAkt-/pMAPK+) cell lines. Univariate and multivariate analysis were performed to evaluate the association of these downstream mainly activated pathways and clinical significance. The mutational status of EGFR and KRAS was determined by direct sequencing.

Results: Of the 99 cases, rates of the Akt pathway type and the MAPK pathway type were 32% (n=32) and 29% (n=29), respectively. Cases with immunoreactivity to both of the antibodies were not included in either type. Mutation assay revealed EGFR mutations in 55 cases (55%), and KRAS mutation in 7 cases (7%). The predictive factors for the MAPK pathway type were cumulative smoking and KRAS mutation by univariate analysis. As all the cases with KRAS mutations were strongly positive for pMAPK, KRAS mutation status was mostly a highly predictive factor for the Akt pathway type.

Conclusions: Association between Ras/MAPK pathways activation and KRAS mutations was confirmed in tumor specimens of lung adenocarcinoma. The MAPK pathway type was statistically associated also with cumulataive smoking.

P3-007 **BSTB: Molecular Pathology Posters, Wed, Sept 5 – Thur, Sept 6**

Relationship between expression of ERCC1and estrogen receptor in patients with adenocarcinoma of the lung

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Background: The incidence of lung adenocarcinoma in Korea is increasing recently. Estrogen and EGFR pathway may be important in lung cancer carcinogenesis. This study was conducted to investigate the relationship among expression rate of estrogen receptor(ER), ERCC1, Kras and EGFR mutation in adenocarcinoma of the lung.

Material and Method: We performed the retrospective review of clinical information that diagnosed as adenocarcinoma of lung at Kosin University Gospel Hospital, Busan, South Korea from January 1999 to September 2005. Patient's demographics, stage, serum tumor markers, pathologic classification were analyzed. EGFR mutations in exons: 18, 19, 20, 21, and K-ras mutation in exon 12, 13 were analyzed by DNA sequencing. The prevalence of ERα, ERβ, and ERCC1 were assessed by standard immuonohistochemical (IHC) methods using formalin-fixed, paraffin-embeded tumor specimens.

Results: For all 84 patients with adenocarcinoma (stage IA in 10, IB 22, IIA 6, IIB 7, IIIA 25, IIIB 7, and IV 7), 43 (51.2%) were male, 41 (48.8%) female, and mean age was 57 years old. Mutations of EGFR mutations and KRAS were present in 25.7%, 9.5% of samples, respectively. In IHC stain, ER expression was 65.5%, ERα expression in 41.7%, ERβ in 51.2%, and both expressions in 27.4%. The expression rate of ERCC1 was 31%. There were correlation between ER expression and ERCC1 expression especially in ERβ expression (p=0.000). However there was no correlation between ER expression and EFGR